Trinity Delta

Scancell

Outlook

Reasons to be cheerful

11 May 2020

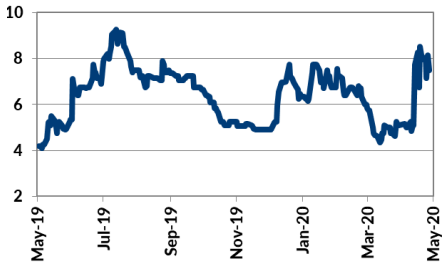
Scancell has two promising vaccine platforms, ImmunoBody and Moditope, that have the potential to treat many cancers, either as monotherapy or in combination with checkpoint inhibitors. The leading ImmunoBody programme, SCIB1, is in a combination Phase II study for metastatic melanoma. Moditope is also expected to enter the clinic, with a Modi-1 Phase I/II trial expected to start during 2021. A third platform, AvidiMab, antibodies that target glycans, can be highly specific to tumour cells and has generated significant industry interest. The expertise in inducing potent immune responses is now also being directed towards a potential COVID-19 vaccine. We value Scancell, using a risk adjusted DCF model, at £72.4m, or 15.6p a share.

Year-end: April 30	2018	2019	2020E	2021E
Sales (£m)	0.0	0.0	0.0	0.0
Adj. PBT (£m)	(4.9)	(6.7)	(6.8)	(7.6)
Net Income (£m)	(4.2)	(5.6)	(5.7)	(6.4)
EPS (p)	(1.3)	(1.5)	(1.2)	(1.4)
Cash (£m)	10.3	4.6	3.5	7.0*
EBITDA (£m)	(4.9)	(6.7)	(6.8)	(7.7)

Source: Trinity Delta Note: Adjusted numbers exclude exceptionals; * FY21 Cash includes a capital increase of £10m

- **Therapeutic vaccines back in vogue** The success of checkpoint inhibitors highlights the need for complimentary techniques to enhance the immune response to a tumour. ImmunoBody and Moditope are ingenious vaccines that elicit consistently strong and sustained cytotoxic effects through potent activation of CD8 and CD4 T cells respectively. ImmunoBody, the most advanced, has demonstrated encouraging high avidity cytotoxic responses in a monotherapy Phase I/II melanoma study.
- **UK/US Phase II combination trial underway** Both the FDA and UK MHRA have cleared the key ImmunoBody programme, SCIB1, to initiate a Phase II combination study. This will examine the tumour response rate, progression-free survival, and overall survival in 25 patients with advanced melanoma. The aim is to explore if combination with a checkpoint inhibitor will improve treatment response. Patient recruitment started in 2019; however, data timings may be impacted by COVID-19.
- **AvidiMab and novel antibodies targeting glycans** Tumour-associated glycans (TaGs) are attractive targets as they are often exquisitely tumour-specific. The challenge has been to produce high affinity antibodies that recognise these small sugars. Three evaluation agreements have been struck in the past six months, highlighting the platform's appeal. This could provide valuable non-dilutive funding.
- **Failing to reflect the strength of the platforms** We value Scancell based on a rNPV and sum-of-the-parts methodology, with conservative assumptions. The valuation is £72.4m, equivalent to 15.6p a share. There are various likely share catalysts over the coming year; including further AvidiMab collaborations, the SCIB1 UK/US trial being underway, and the first SCIB2 and Moditope studies initiating enrolment.

Price	7.5p
Market Cap	£34.9m
Enterprise Value	£31.4m
Shares in issue	465.4m
12 month range	4.03p-10.0p
Free float	68%
Primary exchange	AIM London
Other exchanges	N/A
Sector	Healthcare
Company Code	SCLP.L
Corporate client	Yes



Company description

Scancell is a clinical-stage immuno-oncology specialist that has three technology platforms. Two flexible therapeutic vaccine platforms are progressing through development. ImmunoBody and Moditope induce high avidity cytotoxic CD8 and CD4 responses, respectively, with the potential to treat various cancers.

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Investment case

Three distinct, and attractive, technology platforms

Scancell is a clinical-stage immuno-oncology specialist. It was founded in 1997 as a spin-out of research led by Professor Lindy Durrant at the University of Nottingham. There are now three technology platforms that are clearly distinct: ImmunoBody employs CD8 T-cell pathways; Moditope effects are mediated via CD4 pathways; and the AvidiMab glycans platform, acquired in 2018, consists of specialised monoclonal antibodies. All platforms should have broad applicability in many forms of solid tumours. In May 20 the company announced that its plans to use it expertise in cancer vaccines to develop a potent vaccine against COVID-19. Scancell initially listed on PLUS in 2008 and moved to AIM in 2010. A modest £41m has been raised in equity since inception with £3.9m raised in the past year. The leading shareholders are Vulpes (17.3%) and Calculus Capital (10.7%). The company is based in Oxford and Nottingham, and has 24 employees.

Valuation

Conservative approach gives a value of £72.4m (15.6p a share)

We value Scancell using an rNPV of the three lead indications from the two vaccine platforms, which are then netted out against the cost of running the business and net cash. The success probabilities in each known indication are based on standard industry criteria, but flexed to reflect their differing characteristics. We have used conservative assumptions throughout; erring on the cautious side for factors such as the timing of clinical studies, market launches, adoption curves, and patient penetration. Similarly, we do not attach any value to the AvidiMab platform or COVID-19 vaccine programme as yet. Despite this cautious approach, we value Scancell at £72.4m, equivalent to 15.6p per share.

Financials

Funding required to maximise the vaccine platforms' potential

Scancell ended H120 (October 2019) with a cash balance of £5.79m (vs £7.58m H119) following an operating loss of £3.09m (vs £3.68m H119). £3.83m (net) was raised in May 2018 when Vulpes IM acquired 77.6m new shares at 5p/share. The cash runway is expected to last into Q420. Funding is likely through an equity raise or a meaningful collaboration. We believe Scancell is under-resourced to pursue the opportunities it has. Meaningful AvidiMab deals could provide useful non-dilutive funding, but, in our view, an equity raise would benefit all parties.

Sensitivities

Typical industry risks apply, but amplified by the platform novelty

Scancell's therapeutic vaccine programmes are at the cutting edge of immuno-oncology and, inevitably, carry a higher risk profile. The area of immuno-oncology is increasingly crowded and competitive, with multiple players (ranging from large pharmaceutical groups to biotech companies and even well-funded academic centres) vying to develop the definitive break-through. Equally, the usual industry risks associated with clinical trial results, navigating regulatory hurdles, ensuring sufficient financing is in place, partnering discussions and, eventually, the exit strategy, also apply. COVID-19 has clearly impacted the performance of clinical trials across the industry, and Scancell will not be immune to a degree of delay in patient recruitment and data presentation.

Scancell: advances lead to improved outlook

Scancell has endured a difficult journey. Its promising vaccine platforms were overlooked as immuno-oncology research focussed on checkpoint inhibition. However, treatment limitations mean that the development cycle has turned; the search is now on for complementary, and effective, therapies for use in combination regimens. Vaccines are back in vogue as their ability to potently prime the immune system could compliment checkpoint inhibition perfectly. Scancell is well placed in this now emerging environment, with three distinct technology platforms. We view the latest, AvidiMab, as a potential source of meaningful, non-dilutive funding for the key ImmunoBody and Moditope vaccine platforms. The COVID-19 vaccine programme adds an opportunity to showcase Scancell's expertise. Whilst not without risks, we believe the valuation fails to reflect the opportunities. We value Scancell at £72.4m or 15.6p/share.

Checkpoint inhibitor success brings therapeutic vaccines back into the limelight

Checkpoint inhibitors (CPIs) have transformed the treatment and outlook of many solid tumours; yet, for a variety of reasons, the majority of patients fail to gain long term benefit. This has led to extensive development work on seeking combination therapies that broaden and amplify an appropriate immune response. This has brought therapeutic vaccines back into the spotlight as a means to generate high-avidity T cells that can be used along with CPIs to remove the brakes and so unleash the full potential of a vigorous T cell response.

ImmunoBody, the most advanced, has promising Phase I/II clinical data

ImmunoBody is the most clinically advanced of Scancell's three technology platforms. ImmunoBody vaccines have an elegant design that targets dendritic cells. They achieve efficient direct and cross-presentation of specific epitopes with a consistently strong anti-tumour immune response. Promising and sustained activity was seen in a SCIB1 monotherapy Phase I/II melanoma study in a resected patient population, but for metastatic disease the real potential is in combination with checkpoint inhibitors. A 25-patient Phase II study of SCIB1 in combination with pembrolizumab (Keytruda) in inoperable melanoma is open for patient recruitment in the UK and will also extend to the US following the FDA's IND approval. A second study is planned, with SCIB2, using a liposomal formulation for solid tumours, which will be conducted by Cancer Research UK.

Moditope has a novel mode of action, which appears to be potent and versatile

The **Moditope** platform is unique. Unlike existing cancer vaccine technologies, it is characterised by the induction of CD4 cytotoxic T cells. It exploits the fact that most cancer cells live in stress conditions and to survive often undergo autophagy. This results in modifications such as citrullination and homocitrullination, which initiates an immune cascade that sees CD4 T cells killing tumour cells. The first Moditope product, Modi-1, is expected to start a Phase I/II trial during 2020 in solid tumour indication such as TNBC, ovarian, renal, and head & neck cancers.

AvidiMab platform could be a near-term value generator

The **AvidiMab** platform enhances the therapeutic properties of antibodies, including Scancell's panel of specialised monoclonal antibodies that selectively bind to glycans (carbohydrate elements on proteins or lipids). Tumour-associated glycans (TaGs) are an attractive, but virtually untapped, pool of targets as they are often highly tumour-specific. Application of AvidiMab to these antibodies enhances their ability to kill tumours directly. Three evaluation agreements have already been struck within the past few months, highlighting industry interest, and could lead to more extensive partnering opportunities.

Channeling the immune system against cancers

The immune system is remarkable, but a tumour's various means to overcome it is extraordinary

The body's immune system routinely detects and eliminates abnormal cells through a process known as immunosurveillance. For most of the time this works effectively, but cells can mutate and evolve employing immunosuppressive and evasive mechanisms such that a number escape in a process termed immune editing and a tumour becomes established. Recent work has uncovered many such mechanisms, and in most cases, cancers employ several to avoid recognition and destruction. This has sparked an upsurge of work to develop anti-cancer immunomodulators.

Immuno-oncology is firmly established as mainstream…

Immunotherapy has transformed the treatment regimens for a number of cancer types, with highly promising outcomes for many patients. The most relevant breakthrough has been the introduction of checkpoint inhibitors (CPIs), notably CTLA-4 and PD-1/PD-L1 antagonists such as ipilimumab (BMS's Yervoy), pembrolizumab (Merck's Keytruda), and durvalumab (AstraZeneca's Imfinzi). These CPIs act to reduce the inhibitory mechanisms and so remove the "brake" from the immune system. The results in many cancer types have been remarkable and CPIs have become a cornerstone of various oncology therapies.

…with the goal now to broaden benefit to more patients…

Despite the tremendous progress in the last decade, there is still a frustration that more cancer patients do not benefit from CPIs; it has generally been difficult to increase the proportion of patients who benefit from such treatment to more than c 30%. In part this reflects the heterogeneity within and between cancers, with material variations in mutational load. The classification of tumours according to the level of immune cell infiltration (immunoscore) has begun to help predict how they might respond to different immune based treatments.

…and types of cancer

Tumours can be classified as 'hot' where they have a high number of tumour infiltrating immune cells, or 'cold' where they have limited tumour infiltration. 'Excluded' tumours exhibit immune cells found only in the periphery of the tumour and in the most extreme case 'cold' tumours which are devoid of immune cell infiltrate are referred to as 'immune desert'.

The ideal response needs the "brake" to be removed and the "accelerator" pressed

Typically the cells in a hot tumour have undergone extensive mutation that creates neo-antigens and there is widespread T-cell infiltration. Cancers such as bladder, melanoma, kidney, head and neck, and non-small cell lung cancer are usually hot. In contrast, cold tumours have not yet been infiltrated with T cells, a sign that the immune response is not working for one reason or another. Here the micro-environment usually contains cells that dampen the immune response and inhibit T cells trying to move into the tumour. Most breast cancers, ovarian, prostate, and pancreatic cancer, and glioblastomas are usually cold. It is the lack of T cells that makes it difficult to elicit or provoke an immune response with CPIs.

An understanding of the Cancer Immunity Cycle helps appreciate the complexity

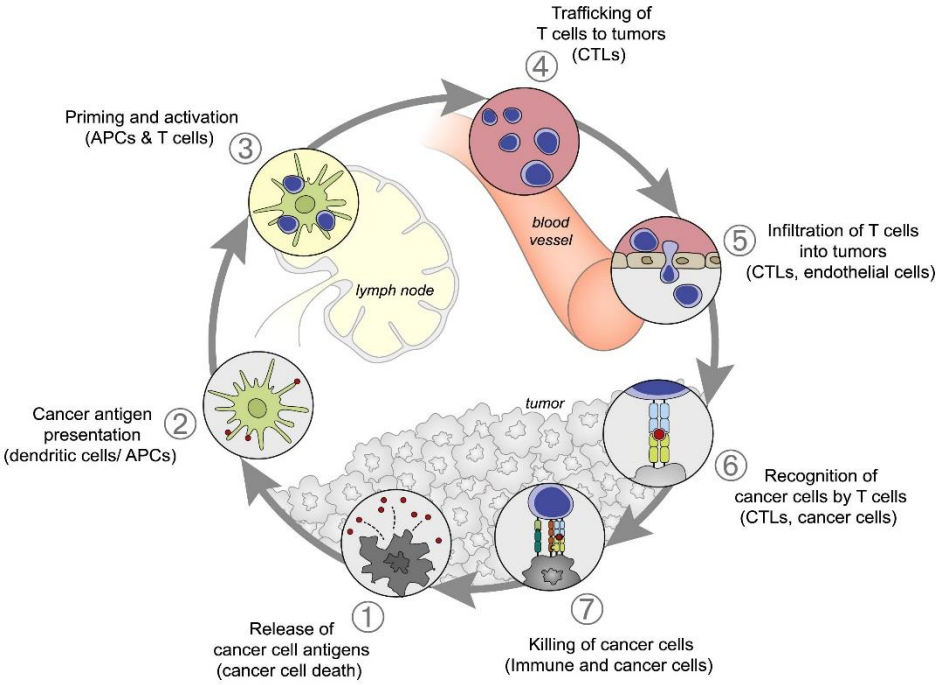
A number of steps are needed for an immune response to lead to effective killing of cancer cells; a series of events must be initiated and allowed to proceed and expand iteratively. This is known as the Cancer-Immunity Cycle (Exhibit 1). Firstly, neoantigens created by oncogenesis or by posttranslational modifications are released and captured by dendritic cells (DCs) for processing (step 1). To yield an anticancer T cell response, this step must be accompanied by signals that specify immunity in case peripheral tolerance to the tumour antigens is induced. Such

immunogenic signals might include pro-inflammatory cytokines and factors released by dying tumour cells.

All steps are critical, but this is "more" critical in determining the final outcomes

Next, DCs present the captured antigens on MHCI and MHCII molecules to T cells (step 2), resulting in the priming and activation of effector T cell responses against the cancer-specific antigens (step 3) that are viewed as foreign or against which central tolerance has been incomplete. The nature of the immune response is defined at this stage, with a critical balance representing the ratio of T effector cells versus T regulatory cells being critical determinants of the final outcome.

Exhibit 1: The Cancer Immunity Cycle



Source: Chen DS et al. Oncology Meets Immunology: The Cancer-Immunity Cycle. Immunity, Volume 39, Issue 1, 1-10.

The difference between a vicious spiral and a virtuous circle is here

The activated effector T cells traffic to (step 4) and infiltrate the tumour bed (step 5), specifically recognize and bind to cancer cells through the interaction between its T cell receptor (TCR) and its cognate antigen bound to MHCI (step 6), and kill their target cancer cell (step 7). Killing of the cancer cell releases additional tumour-associated antigens (step 1 again) to increase the breadth and depth of the response in subsequent revolutions of the cycle.

So many points of contact that can have material consequences…

In cancer patients, the Cancer Immunity Cycle does not perform optimally. For example, tumour antigens may not be detected; DCs and T cells may treat antigens as self rather than foreign thereby creating T regulatory cell responses rather than effector responses; T cells may not properly home to tumours; may be inhibited from infiltrating the tumour; or (most importantly) factors in the tumour microenvironment might suppress those effector cells that are produced.

…but an appropriate effect/harm balance needs to be found

The goal of cancer immunotherapy is to initiate or reinitiate a self-sustaining cycle of cancer immunity, enabling it to amplify and propagate, but not so much as to generate unrestrained autoimmune inflammatory responses. Amplifying the entire cycle may provide anticancer activity, but at the potential cost of unwanted

damage to normal cells and tissues. The challenge is to direct a potent immune response against a tumour, while having a manageable tolerability profile.

Effective and synergistic activity cannot come at the cost of excessive side-effects

As an example, with melanoma, which is the most immunogenic tumour, it has been possible to increase long-term survival to c 60% by combining PD-1 and CTLA-4 antibodies (nivolumab/pembrolizumab and ipilimumab). However, this is associated with a very high level of serious adverse events (Grade 3/4); for instance, in the CHECKMATE-067 Phase III trial, 72% of patients receiving nivolumab and ipilimumab experienced such events compared to 44% in the nivolumab monotherapy arm. Hence, the focus is clearly on identifying combination regimens that will boost overall efficacy and limit treatment resistance, but do so with manageable side-effects.

Judicious use of the "accelerator" whilst timely removal of the "brake" is called for

Because CPIs work by removing brakes on the immune system rather than directly boosting immune function, patients may also benefit from combination therapies that include immunostimulatory substances. It is against this background that interest in therapeutic vaccines has seen a resurgence. The vaccination induces more effective tumour-specific T cell responses, which should synergize potently with CPIs. The goal is to generate a better immune response with the vaccine (or other therapy) and then to remove the suppressive effect of the tumour microenvironment with CPIs (or other immune modulators).

Vaccination has had mixed results historically but now back in vogue

Vaccination is well-established for the prevention of diseases. It has proven to be particularly effective as prophylactic treatments against various viruses in reducing, and even eradicating, diseases. Prophylactic vaccines against HPV (Merck's Gardasil and GSK's Cervarix) have been used widely to prevent women developing cervical cancer, which is caused by the HPV virus. However, progress with the development of therapeutic vaccines, to stimulate a person's immune system to attack their cancer, has previously proved disappointing with the positive results seen in early clinical studies not evident in pivotal Phase III trials. Exhibit 2 highlights some of the reasons why previous efforts may have failed.

Exhibit 2: Potential reasons for a lack of efficacy with therapeutic vaccines

Reason for limited efficacy	Explanation
Epitope recognised as self	Self-antigens normally result in an immune response with a moderate avidity and limited activity, due to negative selection of high avidity T-cells in the thymus.
Use of whole proteins	The use of whole proteins can give rise to a broader T-cell response, compared to the use of peptides; however, most of the epitopes from the whole protein will be self-antigens, which will not result in a high avidity response. Alternatively, immunodominance can occur, resulting in a T-cell response against a small number of epitopes, which might not be the correct ones for anti-tumour efficacy.
Repertoire	Despite the diversity and breadth of epitopes that different TCRs can recognise, it is finite and there are some epitopes to which TCRs tend not to bind.
Delivery system – viral system	Viral delivery systems, such as MVA, can act as potent adjuvants, however the patient might develop a response against the virus rather than the protein/epitope of interest.
Delivery system – depot delivery	A depot delivery system can induce a strong immune reaction, however the depot can act as a sink for the induced T-cell response.
Single-antigen vaccination	Not all tumours express the same antigens, and there is intra-tumour heterogeneity, so few patients might respond if a single antigen is targetted rather than multiple antigens. Similarly, clonal escape (formation of clones of tumour cells that do not express a specific antigen) is likely to be more common with a single- than with multiple-antigen vaccinations.

Source: Trinity Delta

The challenges that need to be overcome are not easy or simple

The three main challenges in developing therapeutic cancer vaccines can be summarised as:

- **low immunogenicity** - tumour cells, which by definition originated from a person's own normal tissues, tend to elicit a low response and the task is to increase the activity of the immune response against them;
- **established disease burden** – to work in the therapeutic setting, vaccine-stimulated immune responses must be able to kill millions or even billions of cancer cells; and
- **immunosuppressive tumour microenvironment** - many potent immunosuppressive mechanisms evolve during the course of cancer progression, which allow tumours to evade destruction.

A high-avidity cytotoxic response is needed and…

To achieve an effective and sustained anti-tumour immune response, it is generally required that high-avidity, cytotoxic T-cells are stimulated. Choosing the right antigen, or epitopes (short amino acid sequences that make up part of the protein), to stimulate an appropriate immune response is the single most important component of cancer vaccine design. Ideally, it/they should be expressed specifically by cancer cells (and not normal cells), present on all cancer cells, be necessary for cancer cell survival (such that the cancer cannot escape immune attack by downregulating the antigen), and be highly immunogenic.

…and selection of the right epitope is critical

The best epitopes are those that are recognised by high avidity T cells, typically tumour-associated antigens (TAA) or neo-antigens. The T cell repertoire to TAA may be restricted due to thymic deletion of T cells in the thymus and many neoepitopes do not produce either better binding to MHC or recognition by T cells. A vaccine approach that presents low amounts of peptide on activated dendiritic cells can only stimulate high avidity T cells no matter the source of antigen. If high avidity T cells are present this approach will be successful, if they are not no immune response will be generated.

ImmunoBody is the most clinically advanced

The first of Scancell's technology platforms, ImmunoBody, is designed to present low amounts of peptide on activated dendritic cells to only stimulate a high avidity cytotoxic CD8 T-cell response against epitopes with very restricted expression patterns.

Moditope has the most exciting preclinical data

The second platform, Moditope, recognises a new class of antigens termed stress induced posttranslational modifications (siPTMs). Moditope products stimulate a cytotoxic CD4 T-cell response, unlike ImmunoBody and other therapeutic vaccines that invoke a cytotoxic CD8 T-cell response. Hence Moditope should be viewed as a totally different class of therapeutic vaccine, targeting a new class of antigens, with the preclinical data suggesting they could have broad applicability for the treatment of multiple cancers.

AvidiMab has the most promising potential for near-term value creation

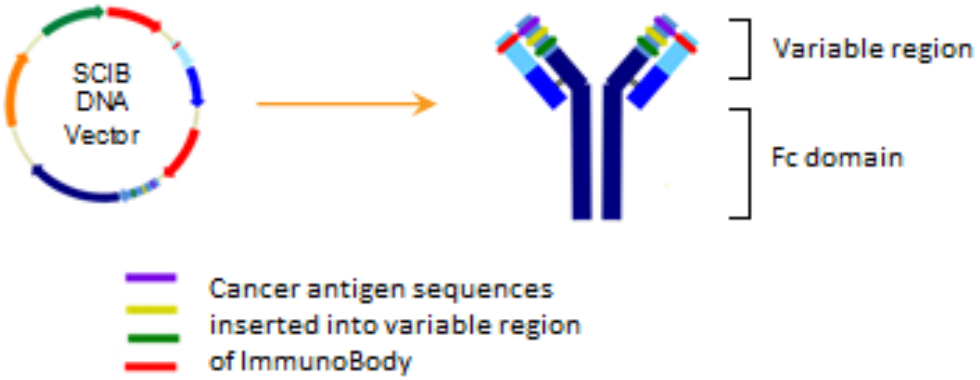
The third platform, AvidiMab, has been applied to a panel of antibodies that target glycans; these are carbohydrates that are attached to proteins or lipids and modify their behaviour. Some glycans are highly specific for tumour cells, known as tumour-associated glycans (TaGs), where they may be expressed at high levels. By selecting TaG targets that are not generally expressed in normal tissues and then producing highly specific antibodies, which can potentially kill tumour cells efficiently both directly and indirectly.

ImmunoBody: the first vaccine platform

An elegant design to produce a broad anti-tumour effect

The ImmunoBody platform is an innovative approach to induce potent cytotoxic CD8 T cell responses against multiple epitopes through a unique dual-mechanism of action. ImmunoBody vaccines have an elegant design to generate high avidity T-cells capable of a broad anti-tumour effect. They are DNA vaccines that encode a human antibody framework, but the parts of the antibody that would normally bind to the target protein, the complementarity determining regions (CDRs), are replaced with carefully selected cytotoxic T lymphocyte (CTL) and helper T cell epitopes from a cancer antigen (Exhibit 3).

Exhibit 3: The structure of the ImmunoBody



Source: Scancell

Therapeutic vaccines require targeting and activation of dendritic cells (DCs) to stimulate both CD4 and CD8 T-cell responses. The ImmunoBody constructs are flexible but the core features include:

- Epitopes selected so they bind to both MHC I (for the CD8 T-cell response) and MHC II (for the CD4 Th-cell response);
- a DNA vaccine with motifs (eg GC rich regions) to ensure it is immunogenic and taken up directly by DCs;
- a Fc region of the protein form that targets activated DCs.

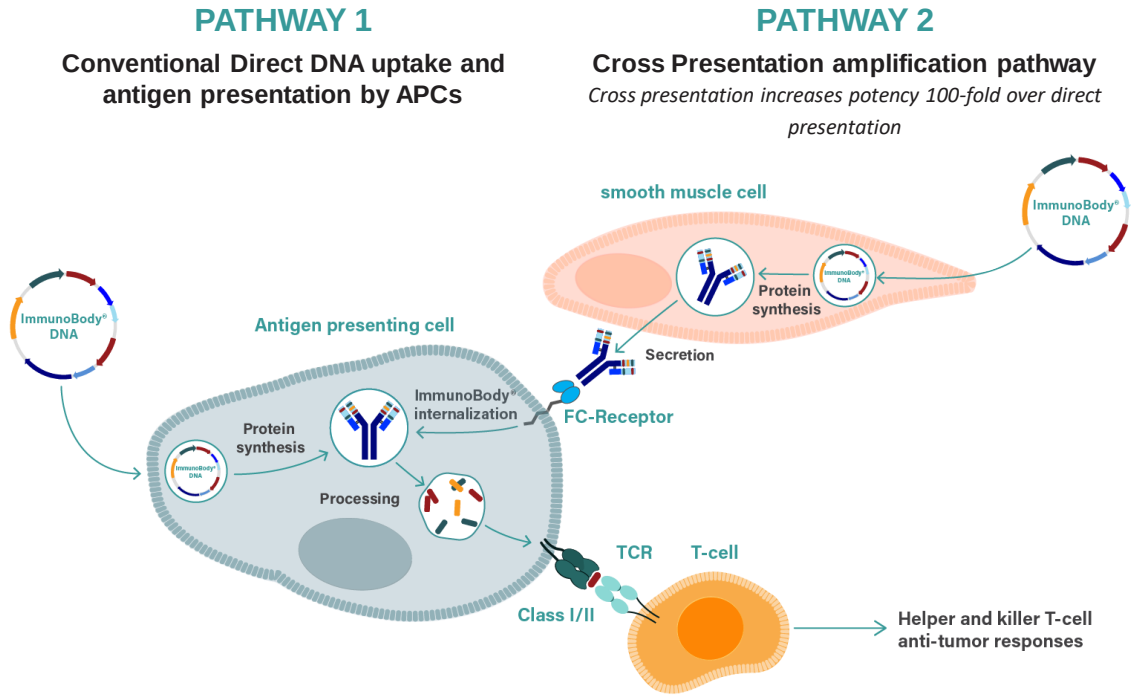
High avidity results from cross presentation via multiple routes

ImmunoBody vaccines activate DCs through two distinctly different and complementary mechanisms that maximise T cell activation and avidity: direct- and indirect/cross-presentation. There are various pathways by which DCs can process antigens, and the highest avidity T-cell response are generated if more than one pathway is used to present the same epitope. In this case, the DNA element is taken up directly by the DCs and the resulting protein is processed directly, whilst an identical protein component is secreted by muscle cells (which is produced at the site of the injection from the DNA) binds to the Fc receptors on DCs leading to the cross presentation (Exhibit 4). Notably, the approach generates both a cytotoxic CD8 cell response and a Th CD4 response.

Should overcome the challenges faced by previous vaccines

The result is that, because of both the direct and cross-presentation, only potent high avidity T cells are generated. This is important since prior vaccine approaches stimulated low avidity T cells that failed to kill tumour cells.

Exhibit 4: The cross-presentation of epitopes by ImmunoBody



Source: Scancell

A potent and targeted cytotoxic response is generated

The ImmunoBody vaccines have been designed so that epitopes for both MHC I and MHC II complexes are produced once they have been broken down by the proteasomes. Epitopes for MHC I are normally 8-11 amino acids in length and generate a CD8 response, and epitopes for MHC II are usually 13-17 amino acids long and result in a CD4 response. The generation of both a Th and Tc cell response is important, as the Tc cells only become activated and able to destroy the tumour cells once Th cells recognise the appropriate epitope and secrete cytokines and chemokines to activate and recruit T cells.

SCIB1 is Scancell's leading clinical programme

Two ImmunoBody vaccines are currently in development:

- SCIB1 targets metastatic melanoma. It incorporates specific epitopes from two proteins, gp100 and TRP-2, which were identified from the cloning of T-cells from patients who achieved spontaneous recovery from melanomas. Both proteins play key roles in the production of melanin in the skin and are expressed by all pigment producing melanoma; and
- SCIB2 targets a variety of solid tumours, including NSCLC (non-small cell lung cancer). It incorporates epitopes from the well-characterised cancer testes antigen, NY-ESO-1, which is normally only expressed in germline cells, and TCR proteins that have been identified to bind to various NY-ESO-1 epitopes.

Results of Phase I trial are highly encouraging

SCIB1 is the lead programme and has completed a dose-escalation Phase I/II monotherapy study in 35 patients with metastatic melanoma. Fifteen patients with tumours received SCIB1 doses of 0.4mg to 8.0mg, whilst 20 fully resected patients received doses of 2mg to 8mg. There was a dose dependent immune response in 88% of patients and an associated anti-tumour effect. Of the fifteen

patients who had tumours present, two patients showed objective reductions in tumour burden and seven achieved stable disease. Of the 20 patients with fully resected disease, 15 were disease-free and were still alive five years post immunisation. The authors concluded that SCIB1 is well tolerated, stimulated potent T cell responses, and the results warranted further evaluation as a single agent adjuvant therapy or in combination with checkpoint inhibitors in advanced melanoma disease.[1]

Clean side-effect profile but delivery device caused issues

There were no serious side-effects associated with SCIB1 therapy. The main adverse event was at the injection site, and was related to the electroporation delivery system, Ichor Medical Systems' TriGrid. This device is able to improve the efficiency of the delivery of DNA vaccines by up to 1000-fold compared to standard needle delivery. It also has an adjuvant effect resulting from local tissue damage and stimulation of pro-inflammatory cytokines.

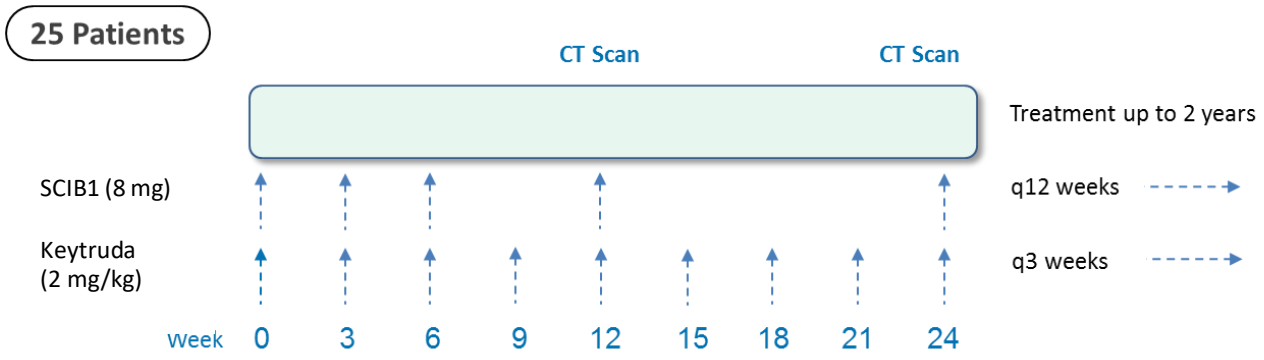
Theoretical synergy with CPIs is backed by strong preclinical data

There is a clear rationale for using an ImmunoBody to prime an immune response against a tumour and enhance the efficacy of CPIs. This potential has been confirmed in preclinical studies; these suggest that SCIB1 and an anti-PD-1 antibody have similar activity when employed as monotherapies (which is consistent with the Phase I/II data), and together have a strong synergistic effect.

Phase I/II trial in combination with pembrolizumab is underway

The SCIB1/pembrolizumab Phase I/II trial is in patients with unresectable stage III/IV melanoma. For stage one of the study, six patients are treated with a primary focus on safety. If the combination therapy has an acceptable tolerability profile, a further 19 patients are treated. The dosing regimen is shown in Exhibit 5, and the trial will be considered a success if ≥12 patients respond to therapy, ie the anti-tumour activity of SCIB1 and pembrolizumab (anti-PD-1) is similar to that seen with the combination therapy of ipilimumab (anti-CTLA-4) and nivolumab (anti-PD-1), but with a better safety profile. Patient recruitment has been initiated in the UK.

Exhibit 5: The trial design of the Phase II study in melanoma with SCIB1 in combination with pembrolizumab



Source: Scancell

FDA clearance of TriGrid 2.0 device is welcomed

The start of the trial was delayed by FDA requesting more information about Ichor's new TriGrid 2.0 electroporation system that is used in this study. This is a newer commercial version of the TriGrid 1.0 device that was employed in earlier

[1] Targeting gp100 and TRP-2 with a DNA vaccine: Incorporating T cell epitopes with a human IgG1 antibody induces potent T cell responses that are associated with favourable clinical outcome in a phase I/II trial PM Patel et al Oncoimmunology. 2018; 7(6): e1433516. Feb 22

clinical work. In February 2020 the FDA cleared the SCIB1 IND (Investigational New Drug) application. The resolution allows Scancell to initiate US site activities and patient enrolment, alongside UK clinical site expansion. We expect these plans to be delayed by the COVID-19 impacts on all such clinical programmes.

SCIB2 addresses a materially larger opportunity

SCIB2 study is organised and funded by CRUK

The first clinical trial with Scancell's second ImmunoBody, SCIB2, is currently being planned with CRUK (Cancer Research UK). The Phase I/II trial is likely to be in non-small cell lung cancer (NSCLC), and unlike with SCIB1, SCIB2 will start clinical development in combination with a checkpoint inhibitor. The study will be performed in the UK (once the COVID-19 restrictions are lifted) and its primary endpoint will be safety and tolerability; however, it will be interesting to see the strength of immune response and the level of tumour response following treatment, especially when viewed against the PD-L1 expression of the individual tumours.

Published preclinical data was highly encouraging

The published preclinical data showed that SCIB2 stimulates higher avidity T cell responses and demonstrated the advantage of combining such T cell responses with checkpoint blockade. When SCIB2 was given together with Treg depletion, CTLA-4 blockade or PD-1 blockade, long-term survival from established tumours was significantly enhanced to 56%, 67% and 100%, respectively. Interestingly, it is the combination of SCIB2 with PD-1 blockade that led to complete tumour regression.

A possible broader utility than SCIB1 but target is less validated

The clinical potential of SCIB2 is considerably greater than that of SCIB1, which only has utility in melanoma and a few other cancers which express gp100 and TRP-2, such as glioblastoma. In contrast, SCIB2 should induce responses against the antigen NY-ESO-1, which is expressed in many different tumours (including sarcomas, neuroblastomas, myeloma, NSCLC, prostate and breast cancers). This suggests that SCIB2 has the capability to be a therapeutic vaccine for many solid tumours and some haematological ones too. It should be noted that there has been much scientific interest in targeting NY-ESO-1, but there is, as yet, limited quantifiable evidence to support its clinical value.

New nanoparticle formulation bypasses need for electroporation

It is also worth highlighting that SCIB2 uses a new lipid nanoparticle formulation. The forthcoming study will administer SCIB2 via a standard injection, rather than using electroporation. The liposomal nanoparticles protect the DNA from degradation and facilitate efficient uptake, expression and T-cell activation against cancer cells. Preclinical studies suggest the nanoparticle formulation is at least comparable to, and could be better than, using electroporation. Successful administration and outcomes should ease future regulatory interactions, help with future study patient recruitment, and provide a useful alternative delivery route for future SCIB development. Additionally, it removes a potential barrier to eventual adoption as no special delivery equipment or training will be required to administer the vaccine in the clinic.

Moditope: a highly innovative approach

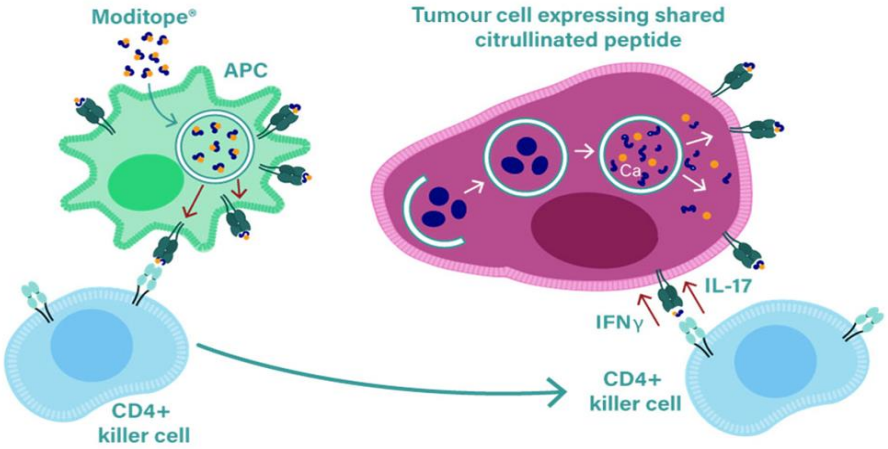
Moditope induces tumour cell destruction via CD4 activation

The Moditope approach is quite different to other therapeutic vaccines in development as it targets a new class of antigens termed siPTMs. There are significant differences between the immune responses generated by Moditope and other therapeutic vaccines, but the most pertinent are the induction of CD4 cytotoxic T-cells and the strength of the anti-tumour response in preclinical studies to date.

A strong and sustained immune anti-cancer response

The mode of action of Moditope vaccines is illustrated in Exhibit 6. Although Moditope is a form of therapeutic vaccine, there are many differences between this and other therapeutic vaccines (including ImmunoBody). A key point of the Moditope approach is that it effectively generates an immune response against the process of autophagy[2], which protects cells experiencing stress.

Exhibit 6: An illustration of the anti-tumour activity of Moditope



Source: Seminars in Immunology VA Brentville 2020; Note: This exhibit uses an example of Moditope that leads to an immune response against cells with citrullinated peptides, but they can also be used to target cells expressing peptides with other modifications.

Exploits the stresses of growing in a tumour's microenvironment

The nature of tumour growth means that most cancer cells live in stressful conditions that are often hypoxic and deficient in nutrients. To survive in this hostile environment, autophagy is required to recycle unwanted proteins and dispose of damaged ones that could become toxic. One siPTM is citrullination, which is caused by activated peptidylarginine deiminase (PAD) enzymes that modify the digested protein fragments within autophagosomes and convert certain arginine residues to citrulline. This alters proteolytic cleavage, generating new epitopes that are presented on MHC-II and stimulate CD4 T cell responses. An alternative second siPTM is homocitrullination (or carbamylation) in which myeloid peroxidase (MPO) converts lysine residues to homocitrulline.

Could break immune tolerance and work in difficult tumours

An hypothesis is that siPTMs allow the immune system to be alerted that cellular stress has occurred, allowing the breaking of self-tolerance and immune recognition. Thus, citrullination in cancer cells creates neo-epitope-like targets for tumour targeting but, unlike *bona-fide* neo-epitopes, would not need to be a form

[2] Autophagy is the normal process that a cell uses to degrade and recycle components of a cell that are damaged or no longer required.

of personalised therapy. Their ubiquity means they could be used to address a range of cancers, notably being of value in tumours with low mutational burden.

Moditope may be effective in many solid tumour types

After administration with Moditope containing citrullinated or homocitrullinated peptides, the peptides are taken up by antigen presenting cells (APCs), such as DCs, that process them and present them by MHC II complexes to CD4 T cells. These primed CD4 T cells infiltrate the tumour microenvironment where they encounter citrullinated/homocitrullinated peptides expressed on the surface of APCs. This results in the CD4 T cells becoming activated and secreting interferon gamma (IFNγ), which induces inflammation. Tumour cells often evade the immune system by creating an anti-inflammatory microenvironment where MHC II expression is not upregulated. However, IFNγ secreted from activated CD4 T cells can shift that balance and induce upregulation of MHC II expression by tumour cells. These active CD4 T cells can now see siPTMs present on MHC II and directly kill the tumour.

Exhibit 7: A comparison of characteristics of Moditope and standard therapeutic vaccines

Reason for limited efficacy	Moditope	Standard therapeutic vaccines
Antigens targeted	Common proteins (eg cytoskeletal proteins) that have post-translational modifications	Tumour-associated antigens or neo-antigens
T-cell response	Cytotoxic CD4 T-cell and CD4 Th cell	Cytotoxic CD8 T-cell and CD4 Th cell
Synergistic with checkpoint inhibitors	Potentially via indirect mechanism	Yes
Delivery system	Intradermal injection	Intradermal, intramuscular or subcutaneous injection

Source: Trinity Delta

Tumour cells may struggle to evade Moditope's actions

Preclinical studies have shown that Moditope has the potential to generate a potent immune response against many tumours. These have also demonstrated that Moditope generates a strong immune memory against the specific modified peptides, as shown by the preclinical studies with a tumour re-challenge assay. Consequently, Moditope vaccines could be used in the adjuvant cancer setting, to reduce the risk that a cancer patient, who has responded well to previous treatment, relapses. The potency of the anti-tumour response seen suggests that tumours have limited defences against an attack from cytotoxic CD4 T-cells, unlike one from cytotoxic CD8 T-cells.

Combination with a CPI could be a compelling proposition

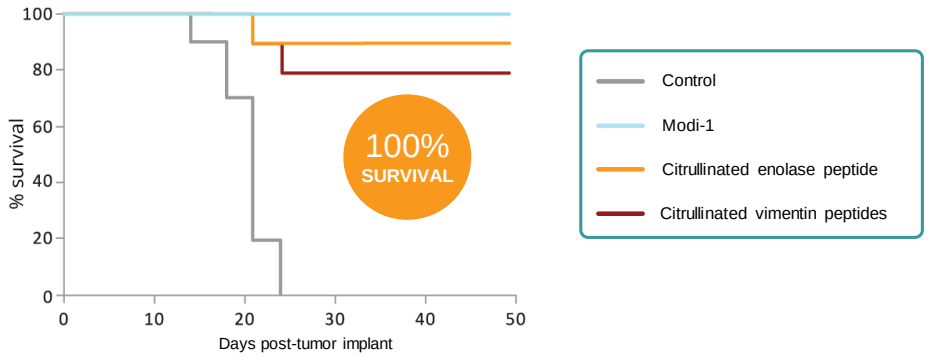
Depending on the results of the preliminary clinical trials with Moditope as a monotherapy, it might be worth investigating the use of Moditope in combination with a CPI. The action of Moditope-induced CD4 T-cells could potentially change the tumour microenvironment, notably through the secretion of IFN-ϒ and the resultant inflammation, thereby converting tumours that are currently considered "cold" into "hot" ones, and therefore become responsive to checkpoint inhibitors and a cytotoxic CD8 T-cell response.

Modi-1: first clinical study being prepared

Modi-1 targets a number of challenging solid tumour types

Modi-1 contains three peptides from two target antigens that are commonly modified in cancer cells. Two are from the cytoskeletal protein, vimentin, which is preferentially digested during autophagy and implicated in tumour metastasis, and one from α-enolase, a key element in many tumours' metabolism pathway. These peptides have been selected and combined to reduce the possibility of tumour escape. These are conjugated to a toll-like receptor (TLR) 1/2 agonist that acts like an adjuvant. Pre-clinical studies in different tumour types, including melanoma, lung, ovarian, pancreatic, and triple negative breast cancer (TNBC), have shown potent T cell responses and a strong anti-tumour activity (Exhibit 8).

Exhibit 8: Preclinical anti-tumour activity of Modi-1 with a melanoma model



Source: Scancell; Note: Modi-1 is a therapeutic vaccine that combines citrullinated enolase peptide and citrullinated vimentin peptides, bound to TLR1/2 agonists to act as adjuvants.

Phase I/II trial will aim to replicate very encouraging preclinical data

A Phase I/II trial programme is being prepared. Two initial cohorts will explore low and high conjugate doses and, if safety and efficacy signals are met, will move into a tumour specific expansion stage looking at TNBC, ovarian, renal, and head & neck cancers. The head & neck cohort will probably include combination therapy with a CPI. The study is expected to start in 2021, but clearly timings will be affected by the COVID-19 impacts on performing clinical trials.

Modi-2 is undergoing preclinical evaluations to optimise targeting

Modi-2 is undergoing preclinical evaluation that is exploring tumour-associated peptide epitopes in which the lysine residues are converted to homocitrulline. At least eight proteins, such as immunoglobulin binding protein (BiP), nucleophosmin (NPM), α-enolase, β-catenin and heat shock protein (HSP-60), are being assessed for their ability to target and mediate a potent anti-tumour effect against many solid cancers (including those with a particularly suppressive microenvironment).

AvidiMab and TaG antibodies: truly novel targets

AvidiMab is an innovative platform in a "hot" new area

It is no longer questioned that monoclonal antibodies have transformed clinical care and patient outcomes, with their extensive use as both therapeutic and diagnostic agents. Almost all target specific peptides or proteins, with few notable exceptions such as dinutuximab (United Therapeutics' Unituxin), which binds to the glycan GD2 and is used to treat children with high-risk neuroblastoma. Yet carbohydrates play many key roles in biology; their presence on proteins can have a major impact on properties such as bioactivity, folding, trafficking, stability, half-life, signalling, trafficking, and mediation of cell–cell interactions.

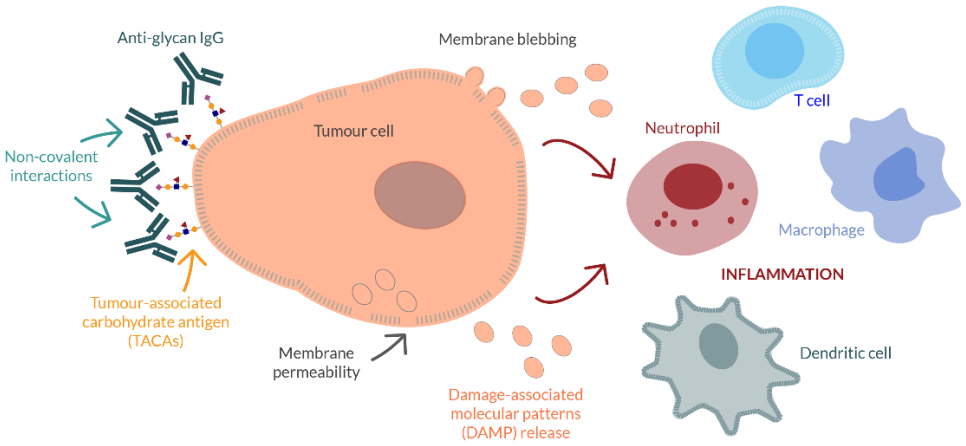
Glycosylation is involved in many aspects of treatment tolerance

Glycosylation is a post-translational modification that occurs inside the cell and results in the addition of sugar motifs, "glycans", to proteins and lipids that are, in most cases, destined for the cell surface. Here they can exert profound effects; for instance, l-selectin on the surface of leukocytes interacts with carbohydrates on endothelial cells to mediate rolling and extravasation from the blood vessel into the surrounding tissue during inflammation. Interestingly, glycosylation is increasingly recognized as a modulator of the malignant phenotype of cancer cells, where the interaction between cells and the tumour microenvironment is altered to facilitate processes such as drug resistance and metastasis.

Attractive targets as selectively found on tumour cells

Glycans are attractive targets for oncology as they are overexpressed in tumours and are essential co-accessory molecules for key cell survival pathways. Blocking these pathways should lead to direct tumour cell killing. Despite the attractiveness of the targets, the challenge has been to produce high affinity monoclonal antibodies that recognise these small sugars. Scancell has developed specialised monoclonal antibodies that selectively bind to tumour associated glycans (TaGs) These are carbohydrate elements on proteins or lipids that have been enhanced with AvidiMab (Exhibit 9). They incorporate specific modifications made to the Fc domain of the antibody, which in turn confers increased avidity and a direct-killing ability. This technology was described in more detail in an Update note (September 2019).

Exhibit 9: AvidiMab selectively targets glycan motifs of tumour cells



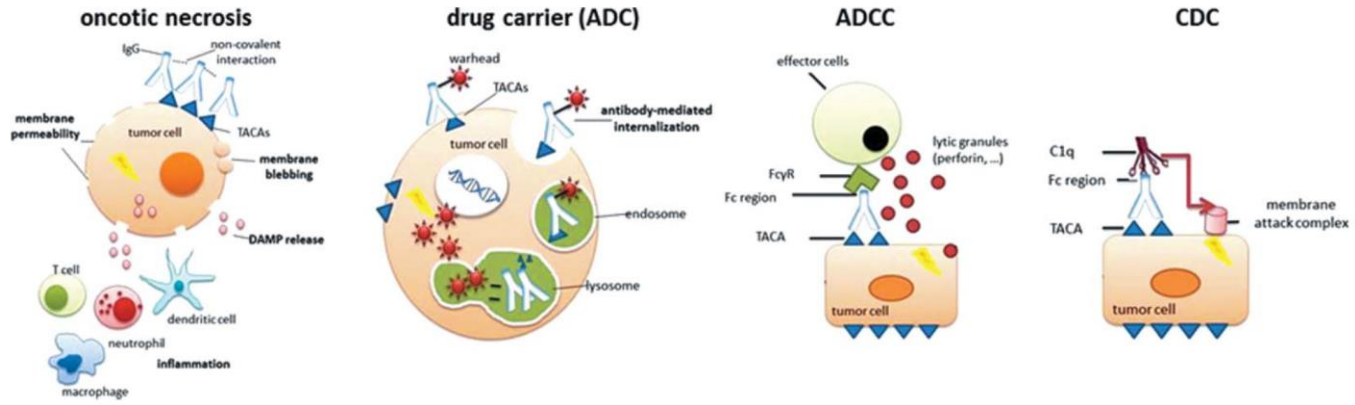
Source: Scancell

A highly flexible approach that can act directly or indirectly

TaGs are an attractive, but virtually untapped, pool of oncology targets as they are often exquisitely tumour-specific. Scancell has produced a series of high affinity monoclonal antibodies that target the TaGs that are highly overexpressed on

cancer cells and which can directly lyse tumour cells without the need for the complement system or immune effector cells. The flexibility of these TaG antibodies means they can be simultaneously used for drug delivery (as antibody drug conjugates ADC), as chimeric antigen receptor T cells (CAR-T), or for redirected T cell killing both directly and indirectly (via ADCC, ADCP or CDC (Exhibit 10).

Exhibit 10: Illustrations detailing the various killing mechanisms of glycan antibodies



Source: Vankemmelbeke M et al; OncoImmunology 5:1; January 2016

Scancell acquired the core intellectual property of the AvidiMab platform and the anti-TaG monoclonal antibodies in April 2018 from Nottingham University. It has since strengthened the data package around both the antibodies and the AvidiMab platform itself, with greater preclinical validation of their potential and additional know-how.

AvidiMab platform offers many attractive features

These antibodies now have many features that make them attractive as potential therapeutic agents, including:

- Sub-nanomolar binding affinities;
- High specificity for target TaG;
- Target TaGs have very limited expression in normal tissues;
- High rates of internalisation for drug delivery;
- Ability to kill tumour cells efficiently (directly or via the immune system).

High level of industry interest, suggests an attractive technology

The TaG antibodies and the AvidiMab technology are clearly becoming a valuable addition to Scancell's key existing technology platforms, ImmunoBody and Moditope. Three research and evaluation agreements have been struck with undisclosed partners since September 2019. Such interest highlights the appeal of the platform to an industry that is actively seeking novel oncology drug targets. Successful initial evaluations could transform these currently non-exclusive agreements into more meaningful partnerships; which, in turn, could provide welcome non-dilutive funding to progress the key development programmes.

COVID-19: developing a long-lasting vaccine

Using proven technology to explore a coronavirus vaccine

Scancell has announced the initiation of a collaborative research programme to develop a vaccine against SARS-CoV-2, the coronavirus that causes COVID-19. While Scancell's primary focus remains on developing its innovative immuno-therapies for cancer, its expertise in developing DNA vaccines that stimulate the body's own immune system has potential application in addressing infection with coronaviruses. The aim is to use the proven clinical expertise in cancer immunology to produce a simple, safe, cost-effective, and scalable vaccine that is able to induce both a durable T cell response and virus neutralising antibodies (VNAbs) against COVID-19.

Goal to produce a better, more potent and versatile vaccine

The current proposal is that Scancell's DNA vaccine will address the SARS-CoV-2 nucleocapsid (N) protein and the key receptor-binding domain of the spike (S) protein to generate both high avidity T cell responses and VNAbs. The N protein is highly conserved amongst coronaviruses; hence it should have the potential to provide protection not only against SARS-CoV-2, but also against future new strains of coronavirus. The objective is not to be the first viable vaccine to reach the market, but to create a vaccine that can produce more potent and longer lasting immune responses. This is particularly important in vulnerable populations with potentially weaker immune systems, such as the elderly and those with relevant underlying health issues.

Initially a three-way collaboration with leading academic centres

The project will initially be a collaboration between the Centre for Research on Global Virus Infections and the Biodiscovery Institute at the University of Nottingham, together with Nottingham Trent University and the John van Geest Cancer Research Centre. Professor Lindy Durrant, Scancell's Chief Scientific Officer and also Professor of Cancer Immunotherapy at the University of Nottingham, will lead the project. Additional development partners are actively being sought, with external funding, including non-dilutive funding from governments and global institutions, also expected. Assuming smooth progress, a Phase 1 clinical trial (likely to be named "COVIDITY") could initiate in Q121.

Sensitivities

Risks are higher than industry average, but upside is greater too

Scancell operates at the cutting edge of immuno-oncology. Clearly, even a modest success would be transformative, but risks inherent in such research are higher than the industry average. The attractiveness of harnessing the body's immune system to treat various tumours has attracted industry-wide attention, with multiple well-funded players operating in a crowded and competitive space. While Scancell's technologies have demonstrable and attractive qualities, an unexpected breakthrough in an unrelated scientific area may sideline its approaches.

A wide array of immuno-oncology approaches are being explored

On the competitive front, both ImmunoBody and Moditope would be complementary to many methods under investigation to enhance the activity of checkpoint inhibitors, such as modulators of tryptophan catabolism and adenosine receptor activity. However, Scancell is also competing directly against other therapeutic vaccine companies, including collaborator BioNTech, and various companies developing oncolytic viruses. This is currently an area of particular interest to big pharma companies.

Industry risks are ever-present, but manageable if understood

More generally, and in common with most innovative healthcare companies, the three main sensitivities relate to the clinical and regulatory aspects, the execution of the commercialisation plans (primarily partnership agreements), and the financial resources required to accomplish these:

- **Clinical aspects:** historic failures of previous therapeutic vaccines cloud expectations of Scancell's programmes. Yet ImmunoBody and Moditope both have different mechanisms of action to any prior attempts and should be judged on their own merits. The design and execution of the clinical programmes is an important determinant of any study outcome, but this is particularly the case in immuno-oncology trials (especially when evaluating differing therapies in combination).
- **Partnership/Licensing and Exit strategies:** the immuno-oncology field is particularly exciting, with many technologies attracting much scientific, and investor, attention. Against such a crowded and "noisy" background, it is always challenging for companies like Scancell to stand out sufficiently to attract the appropriate level of interest from potential partners. However, the BioNTech collaboration and TaG evaluations suggests that good science will be appreciated, and successful innovation rewarded.
- **Financial:** a common refrain is that European biotech companies are seldom financed appropriately to pursue their clinical ambitions in a timely manner. This is arguably true of Scancell, where historically it has lacked the resources to progress its programmes as rapidly as was envisaged. This may yet prove to be a sensitivity in the future.

As with all development-stage companies, COVID-19 may impact Scancell's operations with restrictions on movement and the wider reprioritisation of scientific, clinical, and medical resources potentially causing slippage in timelines.

A sounder financial position would likely benefit all parties

Scancell's shareholder base has long been dominated by smaller investors. Whilst most have been knowledgeable and supportive long term holders, it can be argued that a stronger institutional base could have supported a sounder financial position; with a consequent benefit on development timelines (as above). In that context, we welcome Vulpes Life Sciences Fund joining, in June 2019, Calculus Capital as supportive cornerstone investors.

Valuation

Our rNPV model suggests a value of £72.4m, or 15.6p per share

We value Scancell using an DCF model, where the rNPV of each of the three most advanced oncology projects (adjusted for the likely success probabilities) is summed and netted against the costs of running the operation. The success probabilities are based on standard industry criteria for the respective stage of the clinical development process, but are flexed to reflect the inherent risks of the individual programme, the indication targeted, and the trial design. The key changes to our previous valuation are an update on the cash position and pushing out the launch dates for all the clinical programmes by one year to account for the current uncertainties and potential delays to timelines due to COVID-19.

Exhibit 11: rNPV-based valuation of Scancell

	Total NPV (£m)	Likelihood of success	rNPV (£m)	rNPV/ share (p)	Notes
SCIB1 in melanoma	95.9	20%	17.5	3.8	Peak sales: $325m (£250m) Royalties: 17.5% Launch year: 2025
SCIB2 in NSCLC	195.8	15%	29.4	6.3	Peak sales: $843m (£648m) Royalties: 15% (net of royalties to CRUK) Launch year: 2026
Modi-1 in ovarian cancer, TNBC and head & neck cancer	299.6	10%	26.7	5.7	Peak sales: $1,126m (£867m) Royalties: 17.5% Launch year: 2026
G&A costs	(4.8)		(4.8)	(1.0)	
Net cash	3.5		3.5	0.8	At FY20e
Total	**590.0**		**72.4**	**15.6**	
Discount rate				12.5%	
Exchange rate ($/£)				1.30	
Tax rate				10%	From 2028 with the benefit of UK Patent Box

Source: Trinity Delta

As always, we employ conservative assumptions regarding market sizes and growth rates, net pricing, adoption curves, and peak market penetration. Importantly, we have valued only the clinical programmes (including those ready to enter the clinic) with nothing currently attributed to the technology platforms themselves and their use in other clinical applications. Arguably this is overly conservative, especially as the platforms do have an inherent value. Nonetheless, we would argue that the approach leaves us with ample headroom and scope to revisit our assumptions should the need arise.

Positive news flow could transform Scancell's prospects

Despite such caution, this results in a valuation of £72.4m, or 15.6p per share, for Scancell. There are a number of likely catalysts over the coming year; including further AvidiMab collaborations, the SCIB1 UK/US Phase I/II trial recruiting patients more actively, the first SCIB2 and Moditope Phase I/II studies initiating enrolment, and any increased visibility of progress with the COVID-19 vaccine collaboration. Further out it will the results of these exciting studies that will determine Scancell's outlook. Promising results from any of these trials could transform the company's prospects.

Financials

Solid control over spend has been a key feature

At H120 (31 October 2019) Scancell's cash position was £5.79m (vs £7.58m H119). The operating loss was £3.09m (vs £3.68m H119), with an overall loss of £2.51m (vs £3.24m). The largest expenditures were development costs of £1.98m (up 8% against £1.84m) and administrative expenses of £1.11m (down 38% against £1.83m). The decrease in administrative costs was driven by the comparison with the higher licensing and patent costs for the ImmunoBody and Moditope platforms in the prior period. A net £3.83m was raised in May 2019 when Vulpes IM acquired 77.6m new shares at 5p a share. Vulpes currently owns 17.3% of the shares.

Clinical programmes means cash burn is expected to rise

Looking ahead, for FY20 we expect the operating loss to widen to £6.8m, with a net loss of £5.7m. This is driven by development costs forecast at £4.6m, as clinical programmes start their ramp up. General and administrative expenses are expected to be slightly more modest at £2.2m. For FY21 we expect R&D expenses to rise to £5.6m, but G&A to fall to £2.1m in part reflecting the temporary 25% salary reduction for senior management which will be directed towards funding the initial research work on the COVID-19 vaccine. We forecast a FY21 operating loss of £7.7m and a net loss of £6.4m. The resulting cash outflows mean we are expecting the cash position to be £3.5m at end-FY20 and so are forecasting a funding requirement of c £10m in FY21 (assuming spending on clinical programmes is maintained as planned).

A stronger balance sheet would help maintain focus and progress

This funding requirement may be satisfied, in part at least, through non-dilutive funding such as grants and awards (particularly for the COVID-19 vaccine) or partnership/licensing agreements (especially with AvidiMab). However, we believe that Scancell has suffered historically through having insufficient capital to progress its programmes as rapidly as it should have. In order to not be similarly hampered at such a time-sensitive stage, we would advocate that an equity raise sufficient to ensure financial stability would be advisable. Certainly, management appreciates the size of the commercial opportunity, and has grasped the importance of sensible investment in the clinical programmes and of ensuring the appropriate infrastructure is in place to support them in the very competitive immuno-oncology market. Whilst sensible cost control should remain in place, judicious investment to progress the programmes should be encouraged.

Progress of these novel and differentiated technology platforms, either into or through clinical development, or, in AvidiMab's case, to convert into meaningful collaborations should be key to unlocking shareholder value.

Exhibit 12: Summary of financials

Year-end: April 30	£'000s	2016	2017	2018	2019	2020E	2021E
INCOME STATEMENT							
Revenues		**0**	**0**	**0**	**0**	**0**	**0**
Cost of goods sold		0	0	0	0	0	0
Gross Profit		**0**	**0**	**0**	**0**	**0**	**0**
R&D expenses		(2,009)	(2,766)	(2,855)	(4,152)	(4,633)	(5,560)
General and administrative expenses		(1,034)	(1,783)	(2,087)	(2,577)	(2,202)	(2,100)
Underlying operating profit		**(3,043)**	**(4,549)**	**(4,942)**	**(6,729)**	**(6,835)**	**(7,660)**
Other revenue/expenses		0	0	0	0	0	0
EBITDA		**(3,021)**	**(4,516)**	**(4,914)**	**(6,708)**	**(6,816)**	**(7,640)**
Operating Profit		**(3,043)**	**(4,549)**	**(4,942)**	**(6,729)**	**(6,835)**	**(7,660)**
Interest expense		14	53	3	15	11	7
Profit Before Taxes		**(3,030)**	**(4,495)**	**(4,939)**	**(6,714)**	**(6,824)**	**(7,653)**
Adj. PBT		**(3,030)**	**(4,495)**	**(4,939)**	**(6,714)**	**(6,824)**	**(7,653)**
Current tax income		446	950	745	1,087	1,104	1,279
Cumulative preferred stock dividend		0	0	0	0	0	0
Net Income		**(2,583)**	**(3,545)**	**(4,195)**	**(5,627)**	**(5,720)**	**(6,374)**
EPS (p)		**(1.1)**	**(1.4)**	**(1.3)**	**(1.5)**	**(1.2)**	**(1.4)**
Adj. EPS (p)		**(1.1)**	**(1.4)**	**(1.3)**	**(1.5)**	**(1.2)**	**(1.4)**
DPS (p)		**0.0**	**0.0**	**0.0**	**0.0**	**0.0**	**0.0**
Average no. of shares (m)		227.6	261.6	312.7	387.0	458.5	465.4
Gross margin		*N/A*	*N/A*	*N/A*	*N/A*	*N/A*	*N/A*
BALANCE SHEET							
Current assets		**7,088**	**3,523**	**11,145**	**7,069**	**4,798**	**8,476**
Cash and cash equivalents		6,527	2,672	10,303	4,560	3,543	7,046
Accounts receivable		121	102	97	678	155	155
Inventories		0	0	0	0	0	0
Other current assets		440	749	745	1,831	1,100	1,275
Non-current assets		**3,480**	**3,508**	**3,492**	**3,474**	**3,467**	**3,459**
Property, plant & equipment		65	93	77	59	51	44
Other non-current assets		0	0	0	0	0	0
Current liabilities		**(576)**	**(532)**	**(696)**	**(1,205)**	**(778)**	**(10,778)**
Short-term debt		0	0	0	0	0	(10,000)
Accounts payable		(576)	(532)	(696)	(1,205)	(778)	(778)
Other current liabilities		0	0	0	0	0	0
Non-current liabilities		**0**	**0**	**0**	**0**	**0**	**0**
Long-term debt		0	0	0	0	0	0
Other non-current liabilities		0	0	0	0	0	0
Equity		**9,992**	**6,499**	**13,941**	**9,337**	**7,486**	**1,157**
Share capital		22,047	22,047	33,749	35,026	38,854	38,854
Other		(12,055)	(15,548)	(19,808)	(25,690)	(31,368)	(37,697)
CASH FLOW STATEMENTS							
Operating cash flow		**(2,327)**	**(3,841)**	**(4,060)**	**(7,018)**	**(4,833)**	**(6,483)**
Profit before tax		(3,030)	(4,495)	(4,939)	(6,714)	(6,824)	(7,653)
Non-cash adjustments		44	31	(41)	(248)	50	59
Change in working capital		(12)	(25)	169	(71)	95	0
Interest paid		4	6	3	15	11	7
Taxes paid		667	642	749	0	1,835	1,104
Investing cash flow		**10**	**(14)**	**(11)**	**(3)**	**(12)**	**(13)**
CAPEX on tangible assets		0	(61)	(11)	(3)	(12)	(13)
Other investing cash flows		10	47	0	0	0	0
Financing cash flow		**5,786**	**0**	**11,702**	**1,277**	**3,828**	**10,000**
Proceeds from equity		5,786	0	11,702	1,277	3,828	0
Increase in loans		0	0	0	0	0	10,000
Other financing cash flow		0	0	0	0	0	0
Net increase in cash		**3,468**	**(3,855)**	**7,631**	**(5,743)**	**(1,017)**	**3,504**
Cash at start of year		3,059	6,527	2,672	10,303	4,560	3,543
Cash at end of year		**6,527**	**2,672**	**10,303**	**4,560**	**3,543**	**7,046**
Net cash at end of year		**6,527**	**2,672**	**10,303**	**4,560**	**3,543**	**(2,954)**

Source: Scancell, Trinity Delta Note: Adjusted numbers exclude exceptionals. The short-term debt in FY21 is indicative of the company's funding requirement

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Key personnel

Person	Position	Biography
Dr John Chiplin	Non-Executive Chairman	Joined as Chairman in May 2016. Founder and Managing Director of Newstar Ventures Ltd. Previously CEO of Polynoma, Arana Therapeutics, Geneformatics, and ITI (Intermediary Technology Institute). Non-executive director of many public and private companies. Holds a BPharm (Hons) and PhD from the University of Nottingham.
Dr Cliff Holloway	CEO	Joined as CEO in January 2018. Over 25 years experience of CEO, COO, Business Development roles with Benitec Biopharma, Sienna Cancer Diagnostics, Immune Systems Therapeutics, Biosceptre International, Arana Therapeutics, and Teva Pharmaceuticals Australia. Holds a BPharm (Hons) and a PhD in Medicinal Chemistry from the University of Nottingham.
Professor Lindy Durrant	CSO	Founded Scancell in January 1996 as a spin-out from work she performed at the University of Nottingham (which she joined in December 1983). An internationally recognised tumour immunologist, she is currently Professor of Cancer Immunology at the Department of Clinical Oncology. Over 160 publications in peer-reviewed journals and over 143 patents filed. Holds a BSc (Hons) in Biochemistry and a PhD from Manchester University.

Top shareholdings

	% holding
Vulpes Life Science Fund	17.31
Calculus Capital	10.71
Scancell directors and related holdings	4.12
Top institutional investors	**32.14**
Other shareholders	67.86
Total shareholders	**100.00**

Source: Scancell

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Disclaimer

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